TOXIC EPIDERMAL NECROLYSIS IN THE 90's A BURN CENTER EXPERIENCE
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Toxic epidermal necrolysis (TEN) is an uncommon, severe form of epidermal sloughing with diffuse necrosis of the cutaneous mucosal and epithelial surfaces. The etiology appears to be a hypersensitivity response related to certain infections and medications along with immune system deficiencies. Ten cases of TEN were treated at a regional burn center in the past 2 years. Included were 6 men and 4 women who ranged in age from 29-68 yrs, with an average of 51 years. The mean skin loss was 57% of the TBSA. The mortality rate was 60%. The most common etiology were antibiotics, with sulfa agents comprising 50%. Immunocompromised patients including HIV+ malignancy, and end stage renal disease comprised 60% of the patients and died poorly with a mortality of 83% (5 of 6). Survivors were otherwise healthy individuals that acquired this disease following administration of various medications. Three of nine patients seen at outside hospitals were given steroids prior to transfer. The steroids were tapered and no additional steroids were administered. Treatment included debulking and application of silver nitrate dressings. None of the patients required skin grafting. The average length of hospitalization for survivors was 14.5 days. Our recent experience suggests in patients with severe immune disorders (HIV+, malignancy) presenting with TEN have a poor prognosis. Our results are within reported survival rates for TEN patients with comparable degrees of injury and support early care for these individuals at a burn center.

STUDY OF HYPOXESUSCITATION IN THE RECOVERY OF HEPATIC FUNCTION IN RATS AFTER HYPOVOLEMIC SHOCK B. Chandel, M. Shapiro, R. Hackim, M. Jellinek, A. Ben (USP M. Shapiro),
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Most of the studies have investigated the usefulness of conventional fluid resuscitation in the treatment of hypovolemic shock and recovery of organ function using fluid resuscitation depending on animal body weight. The purpose of this study was to investigate the effectiveness of hypervascularization in the recovery of hepatic function after shock. In SD rats, hypovolemic shock was induced by withdrawing blood to a mean arterial blood pressure of 40
mm of Hg for 90 min, after which blood was reinfused. After 2 hrs of stabilization of hemodynamics, lidocaine (2mg/kg) was injected intravenously as a bolus (Shock Group). Blood samples were drawn at 0, 10, 30, and 60 minutes (T) and hepatocyte function was assessed using Monophosphorylcholine (MEGX) formation kinetics by measurement of MEGX levels (ng/mL). In a separate, shocked group of animals, after return of shed blood, animals either received conventional resuscitation (15ml/kg) or in the form of ringers lactate (CR group) or hypervascularization (30 ml/kg) using ringers lactate (HR group). The control group of animals was unshocked, was not resuscitated, and was injected with lidocaine. MEGX ng/ml (Mean ±SEM)

T CONTROL SHOCK CR HR
0 0.0 0.0 0.0 0.0
6 3.54 ±0.26 11.41 ±4.0 22.09 ±4.46 8.69 ±4.34
10 38.83 ±6.83 43.24 ±3.06 52.64 ±5.79 28.03 ±7.26
60 102.33 ±18.32 57.52 ±4.74 80.08 ±4.02 46.67 ±11.85

Conclusion: Shock produced significant depression (p<0.05) of hepatocyte function which was partially reversed by ringers lactate resuscitation (p<0.05). Hypervascularization significantly depressed hepatocyte function (p<0.05) as compared to conventional resuscitation. Hemodilution and fluid overloading may be responsible for deterioration in hepatocyte function with hypervascularization.

EFFECTS OF VAGAL INNERVATION ON VO2/D02 AND CRITICAL OXYGEN DELIVERY J. D. D. Brown, P. R. Scholter, S. M. King*, J. L. M. Schmoll, S. J. Prot*, P. D. Myerowitz*
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Vagotomy alters regional blood flow distribution by interrupting the tonic inhibitory effect of cardiopulmonary vagal afferent nerves on sympathetic outflow predominantly to the renal, splanchnic, and cutaneous circulations. We hypothesized that the alteration of blood flow distribution by vagotomy would lead to disruption of the oxygen consumption-delivery relationship (VO2/D02) and increase critical DO2 (DO2crit). Nineteen chloralose-anesthetized, paralyzed, splenectomized dogs were submitted to either bilateral vagosympathectomy (n=7), bilateral vagotomy (performed proximal to the nodose ganglion; n=6), or sham operations (n=6) for baseline hemodynamic and cardiopulmonary parameter measurement. VO2 was measured by indirect calorimetry and carbon dioxide blood flow (QCO2) by ultrasonic flow probe. Incremental hemorrhages (1.5 ml/kg) were performed to determine the VO2/D02 relationship and DO2crit. Hemodynamic and cardiorespiratory parameters were measured after each hemorrhage at steady-state VO2. The average DO2crit of the vagosympathectomy group (11.5 +/- 3.2 ml/min/kg) was greater than (p<0.05) both the vagotomy group (9.1 +/- 1.4) and the control group (7.72 +/- 1.2). DO2crit following vagotomy tended to be greater (p>0.05) than control. Qc in the vagosympathectomy group was significantly greater than the other groups. We conclude that vagosympathectomy causes severe disruption of VO2/D02 by disrupting sympathetic nervous control of blood flow to the head and neck. Vagotomy does not have significant effect in this regard.

BURN-INDUCED NEUTROPHIL ADHERENCE TO ISOLATED CARDIAC MYOCYTES J. M. Horton, C. Lin*, B. D. Walker*, R. M. Savidis,
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We have previously shown that burn injury impairs myocyte contractile function. This study was designed to examine the hypothesis that a major thermal injury promotes adhesive interaction of cardiac myocytes with neutrophils (PMN), providing one means by which transendothelial migrating neutrophils directly injure the myocardium. New Zealand White rabbits (2.5-3 kg) were anesthetized (isoﬂurane) and given a full thickness scaldburn over 30% of the total body surface area and resuscitated (lactated Ringer's, 4 cc/kg/hr burn, Parkland formula); sham burn for controls); 24 hr postsburn, rabbits were sacrificed and neutrophils were isolated from anticoagulated blood, yield a 10,000 fold purification with >95% PMN with >95% viability. Cardiac myocytes were isolated by retrograde perfusion of the heart with a Ca2+-free-collagenase-tyrosinase buffer. After enzymatic digestion, myocytes were mechanically disassociated, filtered, washed, and suspended in MEM medium at a concentration of 50,000 cells/ml. Viability was measured (trypan blue dye exclusion) to